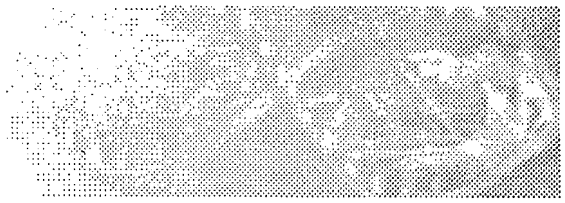
DrugBank



.97 mg/L Solubility

DrugBank
Lansoprazole
(APRD00077)

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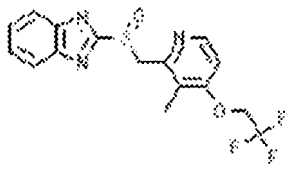
Creation Date	2005/6/27 4:58:37 GMT
Last Update	Feb 01, 2007
Accession Number	APRD00077
Generic Name	Lansoprazole
Brand Names/Synonyms	1. Agopton 2. Amarin 3. Aprazol 4. Bamalite 5. Biuret 6. Biuret Gr 7. Biuret Reagent 8. Biuret Reagent Solution 9. Blason 10. Compraz 11. Dakar 12. Ilsatec 13. Ketian 14. Lancid 15. Lanproton 16. Lansopep 17. Lansoprazol [Inn-Spanish] 18. Lansoprazole [Usan:Ban:Inn] 19. Lansoprazolum [Inn-Latin] 20. Lanston 21. Lanz 22. Lanzol-30 23. Lanzopral 24. Lanzor 25. Lasoprol 26. Limpidex 27. Mesactol 28. Monolitum 29. Ogast 30. Ogastro 31. Opiren 32. Prevacid 33. Prevacid Iv 34. Prevacid Solutab

	35. Prevpac 36. Prezal 37. Pro Ulco 38. Promp 39. Prosogan 40. Suprecid 41. Takepron 42. Ulpax 43. Zoprol 44. Zoton
Brand Name Mixtures	Not Available
Chemical IUPAC Name	2-[[3-methyl-4-(2,2,2-trifluoroethoxy)pyridin-2-yl]methylsulfinyl]-1H-benzoimidazole
Chemical Formula	$C_{16}H_{14}F_3N_3O_2S$
Chemical Structure	
CAS Registry Number	103577-45-3
InChi Identifier	InChI=1/C16H14F3N3O2S/c1-10-13(20-7-6-14(10)24-9-16(17,18)19)8-25(23)15-21-11-4-2-3-5-12(11)22-15/h2-7H,8-9H2,1H3,(H,21,22)/f/h21H
KEGG Compound ID	C07067
PubChem ID	Substance: 196229 Compound: 3883
ChEBI ID	Not Available
PharmGKB ID	PA450180
HET ID	Not Available
SwissProt ID	Not Available
GenBank ID	Not Available
Drug ID Number [DIN]	02165503
RxList Link	Lansoprazole
FDA Label	Click for FDA Label (pdf)
Material Safety Data Sheet (MSDS)	Not Available
Synthesis Reference	A. Nohara, Y. Marki, U.S. Pat. 4,628,098 (1986)
Molecular Weight	369.363 g/mol
Melting Point	178-182 °C

H_2O Solubility	0.97 mg/L
State	Solid
LogP/Hydrophobicity	1.733
pKa/Isoelectric Point	Not Available
NMR Spectrum	Not Available
Mass Spectrum	Not Available
MOL File Image	View 2D Structure
MOL File Text	Click Here for MOL File
SDF File	Click Here for SDF File
PDB File Calculated Image	View 3D Structure
PDB File Calculated Text	Click Here for PDB File
PDB Experimental ID	Not Available
Smiles String	CC1=C(C=CN=C1CS(=O)C2=NC3=CC=CC=C3N2)OCC(F)(F)F
Drug Type	Approved Drug
Drug Category	■ Anti-ulcer Agents ■ Anti-Infectives ■ Proton-pump Inhibitors ■ ATC:A02BC03
Indication	For treatment of Acid-reflux disorders (GERD), peptic Ulcer Disease, duodenal ulcers, esopha Ellison syndrome
Pharmacology	Lansoprazole, an acid proton-pump inhibitor similar to omeprazole, is used as an untiulcer dru and maintenance of healing of duodenal or gastric ulcers, erosive and reflux esophagitis, NSA Zollinger-Ellison syndrome, and Barrett's esophagus. Lansoprozole is active against *Helicoba* plasma elimination half-life of lansoprazole does not reflect its duration of suppression of gast Thus, the plasma elimination half-life is less than two hours, while the acid inhibitory effect las hours.
Mechanism of Action	Lansoprazole belongs to a class of antisecretory compounds, the substituted benzimidazoles, anticholinergic or histamine H_2-receptor antagonist properties, but rather suppress gastric aci inhibition of the (H^+,K^+)-ATPase enzyme system at the secretory surface of the gastric parieta enzyme system is regarded as the acid (proton) pump within the parietal cell, Lansoprazole h as a gastric acid-pump inhibitor, in that it blocks the final step of acid production. This effect is leads to inhibition of both basal and stimulated gastric acid secretion irrespective of the stimul
Absorption	The absorption of lansoprazole is rapid, with mean C_{max} occurring approximately 1.7 hours a relatively complete with absolute bioavailability over 80%.
Toxicity	Symptoms of overdose include abdominal pain, nausea and diarrhea.
Protein Binding	97%
Biotransformation	Hepatic. Two metabolites have been identified in measurable quantities in plasma (the hydrox sulfone derivatives of lansoprazole). These metabolites have very little or no antisecretory act thought to be transformed into two active species which inhibit acid secretion by (H^+,K^+)-ATP cell canaliculus, but are not present in the systemic circulation.

Half Life	1.5 (± 1.0) hours
Dosage Forms	Enteric coated capsules
Patient Information	Click for Patient Information
Interactions	Click for Interactions
Contraindications	Click for Contraindications
Drug Reference	http://www.drugs.com/cons/Lansoprazole.html http://www.rxlist.com/cgi/generic/lansop.htm
Organisms Affected	Humans and other mammals
Phase 1 Metabolizing Enzyme	CYP2C19
Phase 1 Metabolizing Enzyme Sequence	> sp|P33261|CP2CJ_HUMAN Cytochrome P450 2C19 (EC 1.14.13.80) MDPFVVLVLCLSCLLLLSIWRQSSGRGKLPPGPTPLPVIGNILQIDIKDVSKSLTNLSKI YGPVFTLYFGLERMVVLHGYEVVKEALIDLGEEFSGRGHFPLAERANRGFGIVFSNGKRW KEIRRFSLMTLRNFGMGKRSIEDRVQEEARCLVEELRKTKASPCDPTFILGCAPCNVICS IIFQKRFDYKDQQFLNLMEKLNENIRIVSTPWIQICNNFPTIIDYFPGTHNKLLKNLAFM ESDILEKVKEHQESMDINNPRDFIDCFLIKMEKEKQNQQSEFTIENLVITAADLLGAGTE TTSTTLRYALLLLLKHPEVTAKVQEEIERVVGRNRSPCMQDRGHMPYTDAVVHEVQRYID LIPTSLPHAVTCDVKFRNYLIPKGTTILTSLTSVLHDNKEFPNPEMFDPRHFLDEGGNFK KSNYFMPFSAGKRICVGEGLARMELFLFLTFILQNFNLKSLIDPKDLDTTPVVNGFASVP PFYQLCFIPV
Phase 1 Metabolizing Enzyme SwissProt ID	CP2CJ_HUMAN

Drug Target 1

Drug Target 1 Name	H+/K+ ATPase (Proton pump)
Drug Target 1 Gene Name	ATP4A
Drug Target 1 Synonyms	1. Potassium-transporting ATPase alpha chain 1 2. EC 3.6.3.10 3. Proton pump 4. Gastric H+/K+ ATPase alpha subunit
Drug Target 1 Protein Sequence	> H+/K+ ATPase (Proton pump) MGKAENYELYSVELGPGPGGDMAAKMSKKKKAGGGGGKRKEKLENMKKEMEINDHQLSVA ELEQKYQTSATKGLSASLAAELLLRDGPNALRPPRGTPEYVKFARQLAGGLQCLMWVAAA ICLIAFAIQASEGDLTTDDNLYLAIALIAVVVVTGCFGYYQEFKSTNIIASFKNLVPQQA TVIRDGDKFQINADQLVVGDLVEMKGGDRVPADIRILAAQGCKVDNSSLTGESEPQTRSP ECTHESPLETRNIAFFSTMCLEGTAQGLVVNTGDRTIIGRIASLASGVENEKTPIAIEIE HFVDIIAGLAILFGATFFIVAMCIGYTFLRAMVFFMAIVVAYVPEGLLATVTVCLSLTAK RLASKNCVVKNLEAVETLGSTSVICSDKTGTLTQNRMTVSHLWFDNHIHTADTTEDQSGQ TFDQSSETWRALCRVLTLCNRAAFKSGQDAVPVPKRIVIGDASETALLKFSELTLGNAMG YRDRFPKVCEIPFNSTNKFQLSIHTLEDPRDPRHLLVMKGAPERVLERCSSILIKGQELP LDEQWREAFQTAYLSLGGLGERVLGFCQLYLNEKDYPPGYAFDVEAMNFPSSGLCFAGLV SMIDPPRATVPDAVLKCRTAGIRVIMVTGDHPITAKAIAASVGIISEGSETVEDIAARLR VPVDQVNRKDARACVINGMQLKDMDPSELVEALRTHPEMVFARTSPQQKLVIVESCQRLG AIVAVTGDGVNDSPALKKADIGVAMGIAGSDAAKNAADMILLDDNFASIVTGVEQGRLIF DNLKKSIAYTLTKNIPELTPYLIYITVSVPLPLGCITILFIELCTDIFPSVSLAYEKAES DIMHLRPRNPKRDRLVNEPLAAYSYFQIGAIQSFAGFTDYFTAMAQEGWFPLLCVGLRAQ WEDHHLQDLQDSYGQEWTFGQRLYQQYTCYTVFFISIEVCQIADVLIRKTRRLSAFQQGF FRNKILVIAIVFQVCIGCFLCYCPGMPNIFNFMPIRFQWWLVPLPYGILIFVYDEIRKLG VRCCPGSWWDQELYY
Drug Target 1 Number of Residues	1035
Drug Target 1 Molecular Weight	113961 g/mol

Drug Target 1 Theoretical pI	5.54
Drug Target 1 GO Classification	>>> Function: monovalent inorganic cation transporter activity Function: hydrolase activity, acting on acid anhydrides, in phosphorus-containing anhydrides Function: pyrophosphatase activity Function: catalytic activity Function: nucleoside-triphosphatase activity Function: hydrolase activity Function: ATPase activity Function: hydrolase activity, acting on acid anhydrides Function: ATPase activity, coupled Function: hydrolase activity, acting on acid anhydrides, catalyzing transmembrane movement Function: carrier activity Function: transporter activity Function: ATPase activity, coupled to transmembrane movement of substances Function: primary active transporter activity Function: ion transporter activity Function: ATPase activity, coupled to transmembrane movement of ions Function: P-P-bond-hydrolysis-driven transporter activity Function: cation transporter activity Function: ATPase activity, coupled to transmembrane movement of ions, phosphorylative me Function: binding Function: nucleotide binding Function: purine nucleotide binding Function: adenyl nucleotide binding Function: ATP binding || >>> Process: monovalent inorganic cation transport Process: metabolism Process: cellular process Process: physiological process Process: cellular physiological process Process: localization Process: cell growth and/or maintenance Process: transport Process: ion transport Process: cation transport || >>> Component: integral to membrane Component: cell Component: membrane
Drug Target 1 General Function	Inorganic ion transport and metabolism
Drug Target 1 Specific Function	Catalyzes the hydrolysis of ATP coupled with the exchange of H(+) and K(+) ions across the p Responsible for acid production in the stomach
Drug Target 1 Pathway	map00190 Oxidative phosphorylation
Drug Target 1 Reaction	ATP + H2O + H+(in) + K+(out) = ADP + phosphate + H+(out) + K+(in)
Drug Target 1 Pfam Domain Function	PF00689 Cation_ATPase_C PF00690 Cation_ATPase_N PF00122 E1-E2_ATPase PF00702 Hydrolase
Drug Target 1 Signals	None
	98-118 142-162 299-318

Drug Target 1 Transmembrane Regions	331-348 783-802 813-833 854-876 929-948 963-981 997-1017
Drug Target 1 Essentiality	Essential
Drug Target 1 GenBank ID Protein	561634
Drug Target 1 SwissProt ID	ATP4A_HUMAN (P20648)
Drug Target 1 PDB ID	Not Available
Drug Target 1 3D Structure Text	Not Available
Drug Target 1 3D Structure Image	Not Available
Drug Target 1 Cellular Location	Integral membrane protein
Drug Target 1 Gene Sequence	> H+/K+ ATPase (Proton pump), 3108 bp ATGGGGAAGGCCGAGAACTATGAGCTCTACTCGGTGGAGCTGGGTCCTGGCCCTGGCGGG GACATGGCTGCCAAGATGAGCAAGAAGAAGAAGGCGGGTGGCGGGGGTGGCAAGAGGAAG GAGAAGCTGGAGAACATGAAGAAGGAGATGGAGATTAACGACCACCAGCTGTCAGTGGCG GAGCTGGAACAGAAATACCAGACCAGTGCCACCAAGGGCCTCTCTGCGAGCCTGGCTGCT GAGCTGCTGCTGCGGGATGGGCCCAACGCACTGCGGCCACCACGGGGCACCCCAGAGTAC GTCAAGTTCGCGAGGCAGCTGGCCGGGGGCCTGCAGTGCCTCATGTGGGTTGCCGCCGCC ATCTGCCTCATCGCCTTTGCCATCCAGGCTAGTGAGGGGGACCTCACCACCGACGACAAT CTGTACCTGGCAATCGCTCTCATTGCTGTGGTTGTCGTCACCGGCTGCTTTGGCTACTAC CAGGAATTCAAGAGCACCAACATCATCGCCAGCTTTAAGAACCTTGTGCCACAGCAAGCC ACTGTCATCCGCGATGGAGACAAATTCCAGATCAACGCTGACCAACTGGTGGTGGGCGAC CTGGTGGAGATGAAAGGTGGGGACAGAGTGCCCGCCGACATCCGCATCCTGGCGGCCCAG GGCTGCAAGGTGGACAACTCCTCGCTGACAGGGGAGTCTGAGCCACAGACCCGCTCACCC GAGTGCACGCACGAGAGCCCTCTGGAGACCCGCAACATCGCCTTCTTCTCCACCATGTGC CTTGAGGGCACCGCGCAGGGCCTGGTGGTGAACACGGGCGACCGCACCATCATTGGGCGC ATCGCATCGCTGGCGTCGGGGGTGGAAAACGAGAAGACACCCATCGCTATCGAGATCGAG CATTTTGTGGACATCATCGCGGGCCTGGCCATTCTCTTCGGTGCCACATTTTTTATTGTG GCCATGTGCATTGGCTACACCTTCCTGCGGGCCATGGTCTTCTTCATGGCCATCGTGGTG GCCTATGTGCCTGAGGGGCTGCTGGCCACTGTCACAGTCTGCCTGTCCCTGACAGCCAAG CGCCTGGCCAGTAAGAACTGCGTGGTCAAGAACCTGGAGGCGGTGGAGACATTGGGCTCC ACTTCGGTGATCTGCTCGGACAAGACAGGGACTCTCACTCAGAACCGCATGACTGTGTCC CATCTTTGGTTTGACAACCACATCCACACAGCTGACACCACGGAAGACCAGTCAGGGCAG ACGTTTGACCAGTCCTCGGAGACGTGGCGGGCGCTGTGCCGGGTGCTCACCCTGTGCAAC CGCGCCGCCTTCAAGTCCGGCCAGGATGCAGTGCCTGTGCCCAAGCGCATCGTGATTGGA GACGCATCGGAGACGGCGCTGCTCAAGTTCTCGGAGCTGACGCTGGGCAACGCCATGGGC TACCGGGACCGCTTCCCAAAAGTCTGCGAGATACCCTTCAACTCCACCAACAAGTTCCAG CTGTCCATACATACGCTGGAGGACCCGCGGGACCCGCGACACTTGCTGGTGATGAAGGGC GCCCCCGAGCGCGTGCTGGAGCGCTGCAGCTCCATCCTTATCAAGGGCCAGGAGCTGCCG CTGGACGAGCAGTGGCGCGAGGCCTTCCAGACCGCCTACCTCAGCCTGGGAGGCCTGGGC GAACGCGTGCTCGGCTTCTGCCAGCTCTACCTGAATGAGAAGGACTACCCGCCTGGCTAT GCCTTCGACGTAGAGGCCATGAACTTTCCATCTAGCGGCCTCTGCTTTGCGGGACTTGTA TCCATGATTGACCCACCCCGGGCCACCGTCCCTGATGCTGTGCTCAAGTGTCGCACCGCA GGCATCCGGGTGATCATGGTAACGGGTGACCACCCCATCACCGCCAAGGCCATTGCAGCC AGTGTGGGCATCATCTCGGAAGGCAGCGAGACAGTGGAGGACATCGCTGCCCGCCTCCGT GTGCCCGTAGACCAGGTTAATCGCAAGGATGCCCGTGCCTGTGTGATCAATGGCATGCAG CTGAAGGACATGGACCCATCGGAACTGGTCGAGGCCCTGCGCACCCACCCCGAGATGGTG TTTGCGCGCACCAGCCCCCAGCAGAAGCTGGTGATCGTGGAGAGCTGCCAGCGGCTGGGT GCGATTGTGGCCGTCACGGGGGATGGTGTGAATGACTCCCCAGCTCTGAAGAAGGCAGAC ATCGGAGTAGCCATGGGCATCGCTGGCTCAGATGCTGCCAAAAATGCAGCTGACATGATC CTGCTGGATGACAACTTTGCCTCCATTGTGACAGGCGTGGAGCAGGGTCGACTGATCTTC GACAACCTGAAGAAGTCTATTGCCTACACATTGACCAAGAACATCCCAGAGCTGACACCC TACCTCATCTACATCACCGTCAGCGTGCCCCTGCCCCTCGGGTGCATCACCATCCTCTTC

	ATCGAACTCTGCACTGACATTTTCCCATCTGTGTCCCTGGCATATGAAAAGGCCGAGAGT GACATCATGCACCTGCGTCCACGCAACCCAAAGCGTGACAGATTGGTCAACGAGCCCCTG GCTGCCTACTCCTACTTCCAGATTGGTGCCATTCAGTCCTTTGCTGGCTTCACTGACTAC TTCACGGCAATGGCCCAGGAGGGCTGGTTCCCACTGCTGTGCGTGGGGCTGCGGGCGCAG TGGGAGGACCACCACCTACAAGATCTGCAGGACAGCTACGGCCAGGAGTGGACATTCGGG CAGCGCCTGTACCAGCAGTACACCTGCTACACCGTGTTCTTCATCAGCATTGAGGTGTGC CAGATCGCCGATGTCCTCATCCGCAAGACGCGCCGTCTCTCTGCCTTCCAGCAAGGCTTC TTCAGGAATAAGATCCTGGTGATCGCCATCGTGTTCCAGGTCTGCATCGGCTGCTTCCTG TGCTACTGCCCCGGCATGCCCAACATCTTCAACTTCATGCCCATTCGGTTCCAGTGGTGG CTGGTCCCCCTGCCCTACGGCATCCTCATCTTCGTCTATGATGAGATCCGGAAGCTTGGA GTTCGCTGTTGCCCAGGGAGCTGGTGGGACCAGGAACTCTACTATTAG
Drug Target 1 GenBank ID Gene	J05451
Drug Target 1 Chromosome Location	Chromosome:19
Drug Target 1 Locus	19q13.1

Drug Target 1 SNPs

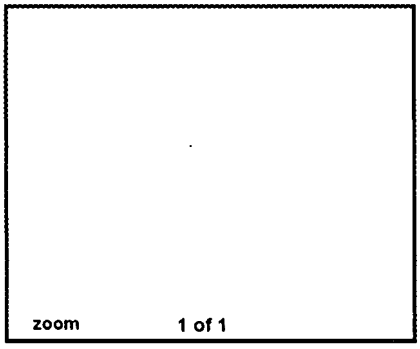
refSNP ID	Function Validation	Alleles Position	Amino Acids Position	Allele Frequencies
rs2733743	nonsynonymous [2+5]	A-G 201	Val[V]-Ala[A] 265	African: A 0 G 1 European: Not Av Asian: A 0.539 G
rs2733739	intron [2+3+4]	A-G 201	Not Available	African: A 0.614 G European: A 0.32 Asian: A 0.272 G
rs2251124	intron [2+4+5]	A-G 501	Not Available	African: G 0.258 A European: G 0.34 Asian: G 0.233 A
rs748213	intron [2+3+4+5]	C-T 194	Not Available	African: C 0.792 T European: C 0.57 Asian: C 0.719 T
rs10416513	intron [1+4]	A-G 470	Not Available	African: G 0.407 A European: G 0.71 Asian: G 0.62 A 0
rs11084823	intron [1+4]	C-T 501	Not Available	African: T 0.833 C European: T 0.85 Asian: T 0.717 C
rs17776451	intron [2]	G-T 101	Not Available	African: G 0.975 T European: G 0.95 Asian: G 0.837 T
rs743541	intron [2+5]	C-G 271	Not Available	African: G 0.908 C European: G 1 C Asian: G 1 C 0
rs2230181	synonymous 5	A-C 151	Ile[I]-Ile[I] 503	Not Available
rs1047217	synonymous [5]	A-G 201	Gln[Q]-Gln[Q] 236	African: G 1 A 0 European: G 1 A Asian: G 1 A 0
rs2854924	synonymous 5	A-C 201	Ile[I]-Ile[I] 503	Not Available
rs10422358	synonymous 5	A-G 201	Phe[F]-Phe[F] 103	Not Available
				African: G 1 T 0

	rs2854923	synonymous [5]	G-T 201	Leu[L]-Leu[L] 402	European: G 1 T Asian: G 1 T 0
	rs12609743	intron 5	G-T 394	Not Available	African: T 0.981 G European: T 0.96 Asian: T 0.952 G
	rs12609727	intron 5	C-T 329	Not Available	African: T 0.958 C European: T 0.92 Asian: T 0.944 C
	rs12977572	intron 5	A-G 201	Not Available	African: G 1 A 0 European: G 0.99 Asian: G 0.971 A
	rs2733740	intron 5	A-C 201	Not Available	African: A 1 C 0 European: A 1 C Asian: A 1 C 0
	rs1044255	synonymous 5	C-T 51	Leu[L]-Leu[L] 198	Not Available
	rs11882238	intron [1+2+4+5]	A-C 318	Not Available	African: C 0.75 A European: C 0.58 Asian: C 0.386 A
	rs1269215	nonsynonymous [5]	C-T 201	His[H]-Tyr[Y] 132	African: T 1 C 0 European: T 1 C Asian: T 1 C 0
	rs2239945	synonymous [1+2+5]	C-T 201	Val[V]-Val[V] 203	African: C 0.983 T European: C 0.83 Asian: C 0.597 T
	rs4442931	synonymous 5	A-G 201	Ser[S]-Ser[S] 196	Not Available
	rs759996	intron [1+2+3+4+5]	C-T 126	Not Available	African: T 1 C 0 European: T 0.83 Asian: T 0.601 C
Drug Target 1 References	2160952 2176086 3036582				

Last Update: Nov 06, 2006

O104 Omeprazole

Sigma **solid**



Synonym	5-Methoxy-2-[[(4-methoxy-3,5-dimethyl-2-pyridinyl)methyl] sulfinyl]-1H-benzimidazole
	Antra
	Losec
Molecular Formula	$C_{17}H_{19}N_3O_3S$
Molecular Weight	345.42
CAS Number	73590-58-6
MDL number	MFCD00083192

Expand/Collapse All

Price and Availability

Click For Pricing and Availability

Descriptions

Biochem/physiol Actions	Binds covalently to proton pump; inhibits gastric secretion.
Caution	Hygroscopic, photosensitive

Properties

form	solid
color	white
solubility	H_2O: 0.5 mg/mL
	DMSO: >19 mg/mL
	ethanol: 4.5 mg/mL
storage temp.	2-8°C

References

Reference

Morii, M., et al., Different biochemical modes of action of two irreversible H^+K (+)-ATPase inhibitors omeprazole and E3810. *J. Biol. Chem.* **268**, 21553, (1993) abstract

Ritter, M. et al., Effect of inhibitors of Na+/H+-exchange and gastric H^+/K^+ ATPase on cell volume intracellular pH and migration of human polymorphonuclear leucocytes. *Br. J. Pharmacol.* **124**, 627-638, (1998) abstract

Safety

Hazard Codes	Xi
Risk Statements	36/37/38
Safety Statements	26-36
WGK Germany	2
RTECS	DD9087000

Related Categories

... Xenobiotics and Drug Metabolism > **Inhibitors**

... Monovalent Ion Channels > **Potassium Channel Modulators**

... Xenobiotics and Drug Metabolism > **Substrates**

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,256/L

LOSEC (omeprazole magnesium)
10 and 20 mg delayed release tablets

H^+, K^+-ATPase Inhibitor

NOTE: When used in combination with amoxicillin, clarithromycin or metronidazole, the Product Monographs for those agents must be consulted and followed.

PHARMACOLOGY

Omeprazole inhibits the gastric enzyme H^+, K^+-ATPase (the proton pump) which catalyzes the exchange of H^+ and K^+. Omeprazole is effective in the inhibition of both basal acid secretion and stimulated acid secretion. The inhibition is dose-dependent. Daily oral doses of omeprazole 20 mg and higher showed a consistent and effective acid control. Information from clinical trials in patients with duodenal ulcers in remission indicate that LOSEC (omeprazole magnesium) 20 mg tablets demonstrate the same inhibition of stimulated acid secretion and similar effect on 24-hour intragastric pH as LOSEC 20 mg capsules. The mean decrease in peak acid output after pentagastrin stimulation was approximately 70%, after 5 days of dosing with LOSEC 20 mg tablet once daily.

The 20 mg tablet and the 20 mg capsule are not bioequivalent in terms of plasma omeprazole AUC, C_{max} and t_{max}. LOSEC 20 mg tablets demonstrate, after repeated dosing, increased plasma omeprazole AUC (18%) and maximum concentration (41%) in comparison to omeprazole 20 mg given as capsules.

The omeprazole capsule (as a multiple unit formulation) is usually emptied gradually from the stomach into the intestine. In contrast to the capsule, the tablet (as a single unit formulation) will enter the intestine and dissolve as one unit. Consequently, the absorption and first pass metabolism of the tablet take place only during a very limited period. This may be one of the reasons for the difference observed in the pharmacokinetic variables of the two formulations.

LOSEC tablets are absorbed rapidly. Food has no effect on the bioavailability of the tablet. Peak plasma levels occur on average within 2 hours.

LOSEC (omeprazole magnesium) 20 mg tablets and LOSEC 20 mg capsules have an equivalent effect on the inhibition of stimulated acid secretion and on 24-hour intragastric pH.

These data support the conclusion that LOSEC 20 mg tablet and capsule can be used with equivalent efficacy in the treatment of conditions where a reduction of gastric acid secretion is required.

The equivalence of two 10 mg LOSEC (omeprazole magnesium) tablets to one 20 mg LOSEC tablet (omeprazole magnesium) has been demonstrated by a bioequivalence study in healthy volunteers.

The antisecretory effect of omeprazole is directly proportional to the AUC; it is not dependent on the plasma concentration at any given time. Omeprazole is 95% bound to plasma proteins.

Treatment with LOSEC alone has been shown to suppress, but not eradicate *Helicobacter pylori* (*H. pylori*), a bacterium that is strongly associated with acid peptic disease. Approximately, 90 to 100% of patients with duodenal ulcers, and 80% of patients with gastric ulcer, are infected with *H. pylori*. Clinical evidence indicates a synergistic effect between omeprazole and certain antibiotics in achieving eradication of *H. pylori*. Eradication of *H. pylori* is associated with symptom relief, healing of mucosal lesions, decreased rate of duodenal ulcer recurrence and long-term remission of peptic ulcer disease, and reducing the need for prolonged anti-secretory therapy.

There is no statistically significant change in the bioavailability (AUC, C_{max}) of amoxicillin during concomitant treatment with omeprazole, in healthy volunteers.

There is an increase in the bioavailability (AUC) and half-life of omeprazole, and bioavailability (AUC) and C_{max} of clarithromycin, during concomitant administration, in healthy volunteers.

There is no statistically significant change in the bioavailability (AUC, C_{max}) of metronidazole during concomitant treatment with omeprazole, in healthy volunteers.

Omeprazole undergoes first-pass metabolism by the cytochrome P-450 2C19 system, mainly in the liver. Following i.v. administration and oral administration (capsules) of omeprazole, 80% of the dose is recovered as urinary metabolites. The remaining 20% is excreted in the feces.

INDICATIONS

LOSEC (omeprazole magnesium) tablets are indicated in the treatment of conditions where a reduction of gastric acid secretion is required, such as:

- duodenal ulcer;
- gastric ulcer;
- NSAID-associated gastric and duodenal ulcers;
- reflux esophagitis;
- symptomatic gastroesophageal reflux disease (GERD) *i.e.*, heartburn and regurgitation;
- dyspepsia*: a complex of symptoms which may be caused by any of the organic diseases listed above, or upon investigation no identifiable organic cause is found (*i.e.*, functional dyspepsia);
- Zollinger-Ellison syndrome (pathological hypersecretory condition);
- eradication of *H. pylori*.

LOSEC, in combination with clarithromycin and either amoxicillin or metronidazole, is indicated for the treatment of patients with peptic ulcer disease associated with Helicobacter pylori infection. The optimal timing for eradication therapy in patients whose ulcer is not clinically active (*i.e.*, asymptomatic) remains to be determined.

The issue of whether or not eradication of *H. pylori* in patients with NSAID-associated ulcers might have beneficial preventive effects has not yet been settled.

In dyspeptic patients with an *H. pylori* infection, the concurrent gastritis can be healed with appropriate eradication therapy.

* A working definition of dyspepsia would include the presence of epigastric pain/discomfort, with or without heartburn and regurgitation which may be accompanied by nausea, vomiting, bloating, belching, flatulence, early satiety or post-prandial fullness. Symptoms may occur either during the day or throughout the night.

CONTRAINDICATIONS

Hypersensitivity to omeprazole or any of the components of this medication (see PHARMACEUTICAL INFORMATION).

WARNINGS

In the presence of any alarm symptom (*e.g.*, significant unintentional weight loss, recurrent vomiting, dysphagia, haematemesis or melena) and when gastric ulcer is suspected or present, malignancy should be excluded, as treatment may alleviate symptoms and delay diagnosis.

Use in Pregnancy

The safety of omeprazole in pregnancy has not been established. LOSEC tablets should not be administered to pregnant women unless the expected benefits outweigh the potential risks.

Nursing Mothers

It is not known if omeprazole is secreted in human milk. LOSEC tablets should not be given to nursing mothers unless its use is considered essential.

Use in Children

The safety and effectiveness of LOSEC tablets in children have not yet been established.

PRECAUTIONS

Use in the Elderly

Elderly subjects showed increased bioavailability (36%), reduced total plasma clearance (to 250 mL/min) and prolonged (50%) elimination half-life (to 1.0 hour) (data obtained from studies with i.v. administration of omeprazole and oral administration of omeprazole capsules). The daily dose in elderly patients should, as a rule, not exceed 20 mg (see DOSAGE AND ADMINISTRATION).

Patients with Hepatic Insufficiency

Patients with impaired liver function showed a 75% increase in bioavailability, reduced total plasma clearance (to 67 mL/min), and a four-fold prolongation of the elimination half-life (to 2.8 hours) (data obtained from studies with i.v. administration of omeprazole and oral administration of omeprazole capsules). A dose of 20 mg omeprazole capsules given once daily to these patients for 4 weeks was well tolerated, with no accumulation of omeprazole or its metabolites. The daily dose in patients with severe liver disease should, as a rule, not exceed 20 mg (see DOSAGE AND ADMINISTRATION).

Patients with Renal Insufficiency

The disposition of intact omeprazole is unchanged in patients with impaired renal function, and no dose adjustment is needed in these patients (data obtained from studies with i.v. administration of omeprazole and oral administration of omeprazole capsules) (see DOSAGE AND ADMINISTRATION).

Information on the bioavailability of LOSEC 20 mg tablet in elderly patients, in patients with hepatic insufficiency, and in patients with renal insufficiency, as well as information on drug interactions are not currently available.

Carcinogenicity

The rat carcinogenicity study (24 months) revealed a gradual development from gastric ECL-cell hyperplasia to carcinoids at the end of their normal life-span during administration with 14-140 mg/kg/day of omeprazole. No metastasis developed. No carcinoids developed during 18 months' high-dose treatment of mice (14-140 mg/kg/day). Similarly, administration of omeprazole up to 28 mg/kg/day in dogs for 7 years did not cause any carcinoids.

The gastric carcinoids in rats were related to sustained hypergastrinemia secondary to acid inhibition and not to omeprazole per se (see TOXICOLOGY). Similar observations have been made after administration of histamine H_2-receptor blockers and also in partially fundectomized rats.

Short-term treatment and long-term treatment with omeprazole capsules in a limited number of patients for up to 6 years have not resulted in any significant pathological changes in gastric oxyntic endocrine cells.

Drug Interactions

The absorption of some drugs might be altered due to the decreased intragastric acidity. Thus, it can be predicted that the absorption of ketoconazole will decrease during omeprazole treatment, as it does during treatment with other acid secretion inhibitors or antacids.

Omeprazole is metabolized by the cytochrome P-450 system (CYP), mainly in the liver. The pharmacokinetics of the following drugs, which are also metabolized through the cytochrome P-450 system, have been evaluated during concomitant use of omeprazole capsules in humans: aminopyrine, antipyrine, diazepam, phenytoin, warfarin, theophylline, propranolol, metoprolol, lidocaine, quinidine, ethanol, piroxicam, diclofenac and naproxen.

Aminopyrine and Antipyrine: After 14 days' administration of 60 mg omeprazole once daily, the clearance of aminopyrine was reduced by 19%; the clearance of antipyrine was reduced by 14%. After 14 days' administration of 30 mg once daily, no significant changes in clearance were noted.

Diazepam, Warfarin and Phenytoin

As LOSEC is metabolized through cytochrome P-450 2C19, it can alter the metabolism and prolong elimination of diazepam, warfarin (R-warfarin) and phenytoin.

Diazepam: Following repeated dosing with omeprazole 40 mg once daily, the clearance of diazepam was decreased by 54%. The corresponding decrease after omeprazole 20 mg was 26%.

Warfarin: Concomitant administration of omeprazole 20 mg in healthy subjects had no effect on plasma concentrations of the (S)-enantiomer of warfarin, but caused a slight, though statistically significant increase (12%) in the less potent (R)-enantiomer concentrations. A small but statistically significant increase (11%) in the anticoagulant effect of warfarin was also seen. Concomitant treatment with omeprazole 20 mg daily did not change coagulation time in patients on continuous treatment with warfarin.

Phenytoin: Following three weeks' treatment with omeprazole 20 mg once daily, the steady-state plasma levels of phenytoin in epileptic patients already receiving concomitant phenytoin treatment were not significantly affected. Urinary excretion of phenytoin and its main metabolite were also unchanged.

After single intravenous and oral doses of omeprazole capsules 40 mg in young, healthy volunteers, the clearance of phenytoin was decreased by 15-20%, and half-life was prolonged by 20-30%. Following repeated dosing with omeprazole 40 mg once daily, the elimination half-life of phenytoin was increased by 27%. Thus, there appears to be a dose-dependent inhibition of elimination of phenytoin by omeprazole.

Patients receiving phenytoin and warfarin should be monitored to determine if it is necessary to adjust the dosage of these drugs when taken concomitantly with omeprazole.

Results from a range of interaction studies with LOSEC versus other drugs indicate that omeprazole, 20-40 mg given repeatedly, has no influence on other clinically relevant isoforms of CYP, as shown by the lack of metabolic interaction with substrates for CYP 1A2 (caffeine, phenacetin, theophylline), CYP 2C9 (S-warfarin), CYP 2D6 (metoprolol, propranolol), CYP 2E1 (ethanol), and CYP 3A (cyclosporin, lidocaine, quinidine, estradiol).

Theophylline: No effects on oral or i.v. theophylline kinetics have been observed after repeated once-daily doses of 40 mg omeprazole.

Propranolol and Metoprolol: No effects on propranolol kinetics were observed in a steady-state trial with 20 mg of omeprazole daily. Similarly, no effects on steady-state plasma levels of metoprolol were observed after concomitant treatment with 40 mg omeprazole daily.

Lidocaine: No interaction with a single intravenous dose of lidocaine or its active metabolite, MEGX, was found after one week's pre-treatment with omeprazole 40 mg once daily. There

were no interactions between omeprazole and lidocaine or MEGX concerning pharmacokinetic variables.

Quinidine: After one week of omeprazole 40 mg once daily, no effect was observed on the kinetics or pharmacodynamics of quinidine.

Ethanol: There was no significant effect on the pharmacokinetics of ethanol after omeprazole 20 mg.

Piroxicam, Diclofenac and Naproxen: There was no significant effect on the steady-state pharmacokinetics of piroxicam, diclofenac, and naproxen following repeated dosing with omeprazole 20 mg, in healthy volunteers.

No interaction with food after repeated dosing of LOSEC tablets has been found. No interaction with antacids administered concomitantly with omeprazole (given as capsules) has been found.

ADVERSE REACTIONS

Omeprazole is well tolerated. Most adverse reactions have been mild and transient, and have shown no consistent relationship with treatment. Adverse events have been recorded during controlled clinical investigations in 2764 patients exposed to omeprazole (data taken from controlled clinical studies with omeprazole capsules) or reported from routine use. In a controlled clinical trial comparing omeprazole to placebo, the prevalence of adverse events with omeprazole 40 mg once daily was similar to that with placebo. In short-term comparative double-blind studies with histamine H_2-receptor antagonists, there was no significant difference in the prevalence of adverse events between omeprazole capsules and the H_2-receptor antagonists. An extensive evaluation of laboratory variables has not revealed any significant changes during omeprazole treatment which are considered to be clinically important.

The following adverse events (at a rate of more than 1%) have been reported in individuals receiving omeprazole capsules in controlled clinical situations: diarrhea (2.8%); headache (2.6%); flatulence (2.3%); abdominal pain (1.7%); constipation (1.3%); and dizziness/vertigo (1.1%).

In addition, the following adverse events were reported in clinical trials or were reported from routine use:

Skin: Rarely, rash and/or pruritus. In isolated cases photosensitivity, erythema multiforme, Stevens-Johnsons syndrome, toxic epidermal necrolysis (TEN) and alopecia.

Musculoskeletal: In isolated cases arthralgia, muscular weakness and myalgia.

Central and Peripheral Nervous System: Rarely dizziness, paresthesia, somnolence, insomnia and vertigo. In isolated cases reversible mental confusion, agitation, depression and hallucination occurring predominantly in severely ill patients.

Gastrointestinal: Nausea and vomiting. In isolated cases dry mouth, stomatitis and

gastrointestinal candidiasis.

Hepatic: In rare cases, increased liver enzyme levels. In isolated cases encephalopathy in patients with pre-existing severe liver disease, hepatitis with or without jaundice and hepatic failure.

Endocrine: In isolated cases gynecomastia.

Hematologic: In isolated cases, patients have developed leukopenia and thrombocytopenia, agranulocytosis and pancytopenia.

Other: Rarely, malaise. Hypersensitive reactions including urticaria (rarely) and, in isolated cases, angioedema, fever, bronchospasm and interstitial nephritis and anaphylactic shock. In isolated cases increased sweating, peripheral edema, blurred vision, taste disturbances and hyponatraemia.

H. pylori Eradication Combination Therapy: The following adverse events (at a rate of more than 1%) were recorded during controlled clinical trials in 493 patients receiving omeprazole, amoxicillin and clarithromycin: diarrhea (28%), taste disturbances (15%), headache (5%), flatulence (4%), nausea (3%), abdominal pain (2%), ALAT increased (1%), epigastric pain (1%), pharyngitis (1%) and glossitis (1%).

The following adverse events (at a rate of more than 1%) were recorded during controlled clinical trials in 494 patients receiving omeprazole, metronidazole and clarithromycin: taste disturbances (14%), diarrhea (13%), headache (6%), ALAT increased (6%), flatulence (5%), nausea (5%), ASAT increased (5%), dyspepsia (3%), dry mouth (2%), dizziness/vertigo (2%), epigastric pain (1%), pharyngitis (1%), eructation (1%) and fatigue (1%).

Clinical experience with the use of LOSEC 20 mg tablet is limited. In two short term studies (20 mg tablet once daily for a maximum duration of 7 days) in a limited number of patients with duodenal ulcer in remission, the adverse event profile seen with the LOSEC 20 mg tablet is similar to that seen with the LOSEC 20 mg capsule.

SYMPTOMS AND TREATMENT OF OVERDOSAGE

No information is available on the effects of higher doses in man, and specific recommendations for treatment cannot be given. Single oral doses of up to 400 mg of omeprazole capsules have not resulted in any severe symptoms, and no specific treatment has been needed. As in all cases where overdosing is suspected, treatment should be supportive and symptomatic. Any unabsorbed material should be removed from the gastrointestinal tract, and the patient should be carefully monitored.

The oral LD_{50} of omeprazole in male and female rats and mice was greater than 4000 mg/kg. In dogs, the only sign of acute toxicity was vomiting, which occurred at doses of approximately 600 mg/kg (see TOXICOLOGY).

When used in combination with antibiotics, the Prescribing Information/Product Monograph for those antibiotics should be consulted.

DOSAGE AND ADMINISTRATION

Duodenal Ulcer

Acute Therapy: The recommended adult oral dose is 20 mg given once daily. Healing usually occurs within 2 weeks. For patients not healed after this initial course of therapy, an additional 2 weeks of treatment is recommended.

Refractory Patients: In patients with duodenal ulcer refractory to other treatment regimens, the recommended adult doses are 20 mg-40 mg given once daily. Healing is usually achieved within 4 weeks in such patients.

Maintenance Therapy for Duodenal Ulcer: Over 95% of duodenal ulcer patients are *H. pylori*-positive, and should be treated with eradication therapy, as described below. A small percentage of patients who are *H. pylori*-negative will experience a disease recurrence and will require maintenance treatment with an antisecretory agent. The recommended LOSEC dose is 10 mg once daily, increased to 20-40 mg once daily as necessary.

Gastric Ulcer

Acute Therapy: The recommended adult dose is 20 mg given once daily. Healing usually occurs within 4 weeks. For patients not healed after this initial course of therapy, an additional 4 weeks of treatment is recommended.

Refractory Patients: In patients with gastric ulcer refractory to other treatment regimens, the recommended adult dose is 40 mg given once daily. Healing is usually achieved within 8 weeks.

Maintenance Therapy for Gastric Ulcer: About 80% of gastric ulcer patients are *H. pylori*-positive, and should be treated with eradication therapy, as described below. A small percentage of patients who are *H. pylori*-negative will experience a disease recurrence and will require maintenance treatment with an antisecretory agent. The recommended LOSEC dose is 20 mg once daily, increased to 40 mg once daily as necessary.

Reflux Esophagitis

Acute Therapy: The recommended adult dose is 20 mg given once daily. In most patients, healing occurs within 4 weeks. For patients not healed after this initial course of therapy, an additional 4 weeks of treatment is recommended.

Refractory Patients: For patients with reflux esophagitis refractory to other treatment regimens, the recommended adult dose is 40 mg given once daily. Healing is usually achieved within 8 weeks.

Maintenance Therapy for Reflux Esophagitis: For the long-term management of patients with healed reflux esophagitis, 10 mg omeprazole (given as capsules) once daily has been found to be effective in controlled clinical trials of 12 months' duration, and in continuous maintenance treatment, in a limited number of patients, for a period of up to 6 years. Therefore, the recommended adult dose of LOSEC tablets for maintenance treatment of patients with healed reflux esophagitis is 10 mg given once daily. In the case of recurrence, the dose can be

increased to 20-40 mg once daily.

Symptomatic Gastroesophageal Reflux Disease (*i.e.*, Heartburn and Regurgitation)

The recommended adult dose is 20 mg given once daily. Symptom relief should be rapid. If symptom control is not achieved after 4 weeks, further investigation is recommended. Since some patients may respond adequately to 10 mg given once daily, individual dose adjustment can be considered. For the maintenance of symptom relief in patients with gastroesophageal reflux disease (*i.e.*, heartburn and regurgitation) the recommended adult dose is 10 mg given once daily.

NSAID-Associated Gastric or Duodenal Ulcers

The issue of whether or not eradication of *H. pylori* in patients with NSAID-associated ulcers might have beneficial preventive effects has not yet been settled.

Acute Therapy: In patients with NSAID-associated gastric or duodenal ulcers, the recommended adult dose is 20 mg given once daily. Symptom resolution is rapid and healing usually occurs within 4 weeks. For those patients not healed after this initial course of therapy, an additional 4 weeks of treatment is recommended.

Maintenance Therapy: For the prevention of relapse in patients with NSAID-associated gastric or duodenal ulcers, the recommended adult dose is 20 mg given once daily, for up to 6 months.

Dyspepsia

Prior to treating patient presenting with dyspeptic symptoms, it should be determined that these symptoms are originating from the upper gastrointestinal tract. Patients presenting with alarm symptoms (see WARNINGS), and older patients who are at a greater risk of having a serious organic disease, should be investigated prior to the initiation of therapy. If the dyspeptic symptoms are known to be related to a diagnosis of organic disease, the appropriate treatment regimen listed in the sections above should be employed.

If the dyspeptic symptoms are not known to be related to an organic disease, the recommended daily dose of LOSEC is 20 mg once daily for 4 weeks. If after 2 weeks' treatment the patient does not respond to therapy, or there is an early clinical indication of a lack of efficacy, the patient should be thoroughly investigated in order to rule out organic disease (see WARNINGS). If there are indications of a clinical response following the initial 2 weeks of treatment, LOSEC may be continued for an additional 2 weeks. Patients may respond adequately to 10 mg once daily therefore, individual dose adjustment may be considered.

Epigastric pain/discomfort (with or without heartburn and regurgitation) as predominant symptoms, are likely to respond to acid suppression therapy. In all cases, patients who do not respond to 4 weeks' treatment, or whose symptoms recur shortly after discontinuation of treatment, with LOSEC should be investigated for underlying organic diseases.

***Helicobacter pylori* Associated Peptic Ulcer Disease**

Omeprazole, Amoxicillin and Clarithromycin Triple Therapy: The recommended dose for eradication of *H. pylori* is LOSEC 20 mg, amoxicillin 1000 mg and clarithromycin 500 mg, all twice daily for seven days. This dosing regimen can be known as Losec® 1-2-3 A™.

Omeprazole, Metronidazole and Clarithromycin Triple Therapy: The recommended dose for eradication of *H. pylori* is LOSEC 20 mg, metronidazole 500 mg and clarithromycin 250 mg, all twice daily for seven days. This dosing regimen can be known as Losec® 1-2-3 M™.

To ensure healing and/or symptom control, further treatment with 20 mg LOSEC once daily for up to three weeks is recommended for patients with active duodenal ulcer, and with 20-40 mg LOSEC once daily for up to twelve weeks for patients with active gastric ulcer.

Patient compliance with treatment regimens for the eradication of *H. pylori* has been demonstrated to have a positive effect on eradication outcome. In clinical trials, patients treated with triple therapy regimens have shown high compliance rates.

Patients who fail to have their infection eradicated may be considered to have *H. pylori* resistant to the antimicrobials used in the eradication regimen. Therefore, therapy involving alternative effective antimicrobial agents should be considered (if re-treating).

In dyspeptic patients with an *H. pylori* infection, the concurrent gastritis can be healed with appropriate eradication therapy.

Zollinger-Ellison Syndrome

The dose used in the treatment of Zollinger-Ellison syndrome will vary with the individual patient.

The recommended initial dose is 60 mg, given once daily. More than 90% of patients with the severe form of the disease and inadequate response to other therapies have been adequately controlled with doses of 20-120 mg omeprazole capsules daily. With doses greater than 80 mg, the dose should be divided and given twice daily. Doses should be adjusted to the individual patient's need and should continue as long as clinically indicated. Doses up to 120 mg omeprazole capsules three times daily have been administered.

Patients with Renal Insufficiency: No dose adjustment is required (**see PRECAUTIONS**).

Patients with Hepatic Insufficiency: No dose adjustment is required. The daily dose should not exceed 20 mg (**see PRECAUTIONS**).

Elderly Patients: No dose adjustment is required. The daily dose should not exceed 20 mg (**see PRECAUTIONS**).

The tablets should be swallowed whole with sufficient water.

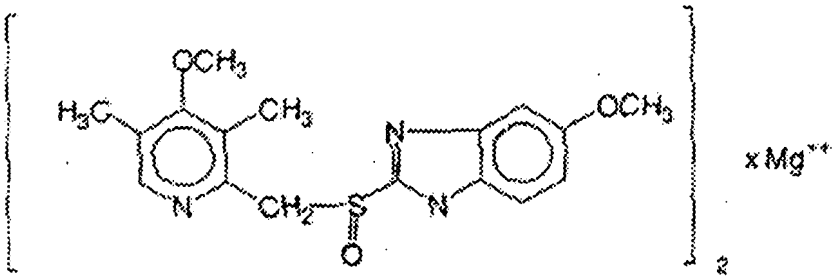
PHARMACEUTICAL INFORMATION

Drug Substance

Proper Name: omeprazole magnesium

Chemical Name: Di(5-methoxy-2-{[(4-methoxy-3,5-dimethyl-2-pyridinyl)methyl]-sulfinyl}-1H-benzimidazol magnesium

Empirical Formula:



$C_{34}H_{36}N_6O_6S_2$ Mg

Molecular Weight: 713.1 (anhydrous basis)

Description: Omeprazole magnesium is a white to off-white crystalline powder, containing between 2 and 4 waters of hydration. The solubility in water is 0.25 g/L, and the solubility in methanol is 10 g/L. The pKa of the benzimidazole (omeprazole base) is 8.8, and that of t pyridinium ion, 4.0.

Composition

Active: omeprazole magnesium anhydrous

mg/tablet: 10.3 (corresponds to 10 mg omeprazole/tablet)
20.6 (corresponds to 20 mg omeprazole/tablet)

Nonmedicinal:
mannitol
microcrystalline cellulose
sodium starch glycolate
hydroxypropyl methylcellulose
talc
sodium stearyl fumarate
methacrylic acid copolymer
polyethylene glycol
titanium dioxide
iron oxide
paraffin

Stability and Storage Recommendations

LOSEC (omeprazole magnesium) tablets are moisture sensitive and are therefore provided in blister compliance packages suitable for direct distribution to the patient. Store in a dry place at controlled room temperature (15-30°C).

AVAILABILITY OF DOSAGE FORMS

LOSEC (omeprazole magnesium) 10 mg tablets are pink, circular and biconvex, printed LOSEC 10 on both sides.

LOSEC (omeprazole magnesium) 20 mg tablets are red-brown, circular and biconvex, printed LOSEC 20 on both sides.

The 10 mg tablets are provided in press-through blister compliance strips in cartons of 28. The 20 mg tablets are provided in press-through blister compliance strips in cartons of 14 and 28, in 10 x 10 unit dose blister packages and in HDPE bottles of 100.

Full Product Monograph available on request.

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